

The management of eclampsia associated with HELLP syndrome in pregnancy

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Abstract

Eclampsia and HELLP (hemolysis, elevated liver enzymes, low platelet count) syndrome represent two major obstetrical emergencies that are associated with important materno-fetal morbidity and mortality rates. The concomitant occurrence of these two entities is rare, and special attention must be offered regarding the individualised case management. We present a case of a 19-years old nullipara, 28 weeks of gestation that was admitted to our clinic with the diagnosis of eclampsia and later developed HELLP syndrome and initially underwent expectative management in a tertiary center of a developing country.

Keywords: : eclampsia, HELLP syndrome, magnesium sulphate

INTRODUCTION

Eclampsia is defined as the occurrence of grand mal seizures during pregnancy or during/after delivery in a woman with preeclampsia, not attributable to other causes. The vast majority of the cases occur in the third trimester, after 28 weeks of gestation [1].

Eclampsia is one of the main causes for maternal mortality in high and low-income countries. The incidence of eclampsia varies between 0.2-0.5 percent of all deliveries according to the World Health Organization's International Collaborative Study of Hypertensive Disorders of Pregnancy [2]. The reported incidence is usually much higher in tertiary referral medical centers and in patients that do not obtain prenatal care.

Several clinical symptoms are suggestive for eclampsia diagnosis: persistent occipital or frontal headaches, photophobia, blurred vision, altered mental status, and epigastric or right upper quadrant pain. In most cases can be seen at least one of these symptoms in 59-75% of the cases [3].

HELLP syndrome is a severe form of preeclampsia-eclampsia and is constituted by hemolysis, elevated liver enzymes and low platelets [4]. The main pathophysiological mechanism encountered in eclampsia and HELLP syndrome represents endothelial dysfunction which plays an important role in the ischemic placental syndrome. The incidence of these two entities together occurs

in 10.8 % to 32.1% [1, 5]. In order to decrease the death rate of eclampsia it is mandatory to establish an early diagnosis and management for HELLP syndrome, to what consider the delivery time of the babies, the prevention of seizure and severe hypertension. The possible complications of preeclampsia are eclampsia, placental abruption, renal failure, coagulopathy, rupture of hepatic hematoma and hypertensive encephalopathy.

We present a case of eclampsia in association with HELLP syndrome that initially underwent expectative management in a tertiary center of a developing country.

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Received December 09, 2019; **Accepted** Mars 2, 2019; Mars 25, 2020

Citation: Tanasă Ingrid-Andrada, Onofriescu¹ M, Tirnovanu Mihaela, Tanase Adina, Ciuntu B, Rosu E., Timofte D., Arcana Stefanita, Toma B.F., Himiniuc Loredana Maria Daniela. The management of eclampsia associated with hellp syndrome in pregnancy. Journal of Surgery [Jurnalul de chirurgie]. 2020; 16 (1): 58-61

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Case report

A 19-years old nulliparous patient, with personal history of tetanic seizures and poor prenatal care was transferred to the Clinical Hospital of Obstetrics-Gynecology „Cuza-Voda” from the Clinical Hospital of Neurosurgery „Nicolae Oblu” from Iasi, Romania, at 29 weeks of gestation with the diagnosis of imminent preterm birth and eclampsia for obstetrical consult and management.

From her admission file we found out that the

patient was previously admitted for inaugural tonic-clonic seizures with generalized onset that happened at home and inside the ambulance that resulted in loss of consciousness, spontaneous emission of feces and urine, tongue biting and postictal confusional state, being treated with intramuscular 5 mg of Diazepam.

On admission to our clinic the patient accused epigastric pain, her blood pressure was 170/110 mmHg and ventricular allure was 135 bpm. Apart from her bitten tongue, the physical examination was normal.

	01.12.2019	01.12.2019	01.12.2019	01.12.2019	02.12.2019	03.12.2019	04.12.2019
CBC							
WBC	30,330	29,190	32,830	20,650	24,530	26,520	23,260
HGB	13,4	15	15	13,4	10,5	9,7	10,2
PLT	320,000	221,000	221,000	201,000	90,000	228,000	209,000
Biochemistry							
TGO	112		481	655	326	23	60
TGP	55	257	312	188	154	16	79
CK			380		3156		
Mg			3	3,2	2,8	1,9	1,9
Bile acids		2		2,9	1,6	1,3	1,9
LDH	675		1689	2212	1124	502	317
Uric acid	11,82		10,39	9,56	9,19	7,72	4,24
CRP			17,55	53,11	77,62	100,96	13,4
Coagulogram							
TQ	15,5	17,1	18,6	15,3	16,4	16	16
INR	1,12	1,26	1,39	1,10	1,11	1,20	1,16
D-DIMERS		6,400-12,800	6,400-12,800	3,200-6,400	1,600-3,200	800-1,600	800-1,600
Urinalysis							
Albumin	300	300	300	300	300	100	300
Microbial flora	numerous	numerous	numerous				

Table I. Chronologically exposure of altered laboratory tests results in our patient, during the hospitalization

The sonographic examination revealed a chronological gestational age of 29 weeks and 4 days, a median gestational age of 27 weeks and 4 days, an estimated fetal weight of 1070 g, normal aspect of amniotic fluid and placenta, the resistance index on mid cerebral artery of 0.82 and 0.79 on umbilical artery.

Her blood tests indicated leukocytosis (30.330/mm³) with neutrophilia (27.390/mm³), normal platelet count (320.000/mm³), hepatocytolysis syndrome (glutamic oxaloacetic transaminase -TGO=112 UI/L, glutamic pyruvic transaminase- TGP= 55 UI/L), hyperuricemia (uric acid- 11.82 mg/dl), increased lactate dehydrogenase- LDH= 675 UI/L, albuminuria 300 mg/day and abundant microbial flora present in urine (table I).

We decided to administer Methyldopa 250 mg, Nifedipine 10 mg, Dexamethasone 6 mg intramuscular and a

perfusion of magnesium sulphate 250 mg/ml in 500 ml of saline. After 3 hours, her blood pressure decreased down to 140/80 mmHg, pulse 120 bpm, normal uterine tonus, she perceived active fetal movements and was continuously monitored in the delivery room. After 5 hours she started to accuse epigastric pain and temporo-spatial disorientation, being transferred to intensive care unit where she developed an eclamptic seizure and was initiated treatment with Magnesium sulphate 2g at 12 hours apart and Diazepam 5 mg intramuscular, with continuous monitoring of blood pressure (143/95 mmHg), pulse 130 bpm, oxygen saturation (92%). Emergency caesarean section was performed under general anaesthesia and a viable male neonate of 900g with Apgar score 3 at 1 minute was delivered. After the surgical intervention her blood pressure was between 140-150/80-90 mmHg and the orotracheal intubation was maintained another 3 hours before opting for sedation.

Six hours after surgery, the patient developed a new eclamptic seizure that required orotracheal intubation (Fentanyl 50 µg/h; Midazolam 2 mg/h; Propofol 40 mg/h), and her blood pressure was 130/80 mmHg, pulse 130 bpm. Clinically, some ecchymosis and petechiae appeared on her upper arms and at venepuncture site and her bloodtests revealed clotting alterations (D-dimers 6,800-12,800 ng/ml, prothrombin time 18.6/s, thrombocytopenia 90,000/mm³) – (table I), so the anaesthesiologist decided to administer 2 mg of NovoSeven intravenously.

In the patient's first post operative day, the Douglas drainage was serosanguinous, counting approximately 400 ml, and the patient received treatment with Cefotaxime 2g bid and Midazolam 1g bid, Methyldopa 250 mg tid and Nifedipine 10 mg bid. In the second day after the caesarean section, the patient was detubated, having a stable status. Moreover, because an inflammatory syndrome indicated by her bloodwork (table I), she received triple antibiotic association with Tienam 1 g tid, Clindamicin 600 mg tid, Fluconazole 400 mg single dose, for 3 days, continued with symptomatic treatment and Amikacin 1 g per day until discharge 3 days later. The neonate remained in the intensive care unit for careful monitoring and management of the possible complications that are associated with prematurity.

Discussions

Eclampsia is one of the leading causes of maternal morbidity and mortality worldwide. Failure of clinicians to predict the life-threatening complications from eclampsia may contribute to rise in maternal mortality due to this disease and its associated complications. The main point in this discussion section is based on an error of case management. We firmly believe that the patient had to be admitted immediately to the intensive care unit and to be offer appropriate magnesium sulphate prophylaxis of the seizures. Only Diazepam was offered to the patient in the ambulance, before reaching our clinic, and the initial dose of Magnesium sulphate received in the delivery room was insufficient for eclamptic seizure prophylaxis. These aspects point out errors of case management, communication between different departments and failure to immediately recognize potential life-threatening complications associated with eclampsia.

In our opinion, proper diagnosis and appropriate management is extremely important in patients with eclampsia, for better maternal as well as perinatal outcome. The neonatal outcome was poor and a proper case management could possibly reduce the neonatal complications associated with prematurity. The newborn remained in the intensive care unit and was carefully monitored in order to manage the complications that appeared.

Clinician must perform a detailed assessment along with thorough history taking (including the symptoms of severe pre-eclampsia) and physical examination. In our case, the ecchymosis and petechiae indicated alterations of coagulation, mainly based on thrombocytopenia. Laboratory tests such as a complete blood count with platelet count, and assessment of serum creatinine and liver enzymes levels, evaluation for urine protein (24-hour collection or protein/creatinine ratio) may aid to establish the diagnosis. Beside these laboratory tests, D-dimers were used in our case for the diagnosis of disseminated intravascular coagulation (DIC).

Maternal complications of severe preeclampsia include pulmonary edema, myocardial infarction, stroke, acute respiratory distress syndrome, coagulopathy, severe renal failure and retinal injury. Our patient had a poor antenatal care and the diagnosis of preeclampsia did not appear in her medical file. A proper management of preeclampsia would have prevented the complications that manifested in the end.

Fetal and newborn complications of severe eclampsia result from exposure to uteroplacental insufficiency or from preterm birth, or both. In our case the newborn complications were due to prematurity.

According to ACOG (2013), for women with severe preeclampsia at or beyond 34 0/7 weeks of gestation, and in those with unstable maternal-fetal conditions irrespective of gestational age, delivery soon after maternal stabilization is recommended [6]. The decision to perform an emergency cesarean section and to deliver the baby at 29 weeks of gestation was made because of maternal status degradation and severe eclamptic seizure that manifested in the intensive care unit and respected this recommendation.

A patient with HELLP Syndrome represents an obstetrical emergency with indication for caesarian section. It is very important to mention the fact that if the patient has previous surgical intervention it exists the possibility to find out adhesions between the intraperitoneal organs. It is interesting to notice that although in other research fields, like technological departments, the adhesion of a thin material film to a biocompatible surface adds positive contributions in the pathophysiology of the disease, in this case, the adhesions can cause a lot of intraoperative difficulties [7-11].

Conclusions

This case report of patient who presented at 29 weeks of pregnancy, with inaugural tonic-clonic seizures with generalized onset that happened at home and in ambulance, epigastric pain, high blood pressure describes a clinical context which associated eclampsia with HELLP syndrome.

She developed complications associated with eclampsia and fortunately had a good obstetric and neurologic outcome, despite of some errors in the patient's management.

Clear protocols for early detection and management of hypertension in pregnancy at all levels of health care are required for better maternal as well as perinatal outcome.

Conflict of Interest

Authors have no conflict of interest to disclose.

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